INFANTILE DIABETIC KETOACIDOSIS: A RARE CASE

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Abstract

Infantile Diabetic Ketoacidosis (DKA) is rare, but most common presentation for diabetes of infancy at onset. DKA frequency vary between 30-75% at diagnosis.[4] We report a case of a 54-day-old infant who came in shock and in severe DKA. She required intubation and paediatric intensive care unit admission. Management was successful where she was out of DKA after 48 hours of admission and eventually able to change to subcutaneous actrapid. Her type of NDM is yet to be categorised as she is still under investigation.

Introduction

Diabetic ketoacidosis (DKA) is one of acute, life threatening diabetic emergency that may complicates diabetes of infancy (Neonatal diabetes mellitus). It is due to severe metabolic derangement produced as a result of insulin deficiency and counterregulatory hormone excess. There will be concomitant hyperglycaemia, cellular starvation and severe depletion of water and electrolytes from both the intra- and extracellular fluid (ECF) compartments.[2,3,6].Overall mortality in children with DKA varies from 0.15% to 0.35% in developed countries and from 3.4% to 13.4% in developing countries.[1]

NDM is a form of monogenic diabetes that manifests during the first 6 months of life. It is a rare form of diabetes, which is estimated to occur in 1 over 20,000 to 500,000 live births.[7,8]DKA is the most common presentation at onset of NDM. [1,4]

We report a case of a 54-day-old infant who presented with DKA and required 16 days of Paediatric Intensive Care Unit(PICU) admission.

Case report

A 54-day-old infant was referred with the diagnosis of severe bronchopneumonia and presumed meningitis. She presented with on and off fever for 3 days with refused in feeding for 1 day. On the day of referral, she cried unconsolably with difficulty in breathing. No history of upper respiratory infection, vomiting or loose stool. Urinary output as usual.

She was delivered full term via spontaneous vaginal delivery with birth weight of 2.5 kg. Mother's antenatal history was unremarkable. There was no family history of diabetes. She was product of non consanguineous marriage.

On presentation to district hospital, the infant who weighed 3.6 kg was lethargic and dehydrated. She was hypothermic with temperature of 35.3'c. Her anterior fontanelle was sunken, pulse was feeble and capillary refill time of 2 seconds. Blood pressure was 95/72 mmhg. She was tachypnoeic, with respiratory rate of 50 per minute. There was recession, but oxygen saturation maintained 99-100 % under nasal prong oxygen. Lung examination was clear. Cardiovascular and respiratory examination were normal. Capillary random blood glucose showed high blood glucose, but blood gas was not taken as it was not available. She was given a total of 20 ml/kg boluses and transported with 7.5% fluid correction to tertiary centre.

On arrival to tertiary centre, the patient was still in shock despite the fluid boluses which showed by her mottled skin, poor skin turgor, capillary refill of 4 seconds and sunken anterior fontanelle. The blood pressure was within range. Her respiratory rate was 40 breaths/min with subcostal and intercostal recession. Her breath was ketotic. She was hyperglycaemic with random blood sugar(RBS) of 41 mmol/L and in severe metabolic acidosis. Venous blood gas revealed pH of 6.818, HCO_3^- of 5 mmol/l, and lactate of 1.8 mmol/l. pCO_2 and paO_2 were 26.8 and 56.9 kPa respectively. Her capillary ketone was 4.3 mmol/l. Diagnosis of DKA was made. She was given another 30 ml/kg fluid boluses and intubated for severe metabolic acidosis. She was placed on intravenous insulin infusion 0.1 unit/kg/hr as per DKA protocol. She was given 10 % correction over 48 hours and was admitted to paediatric intensive care unit(PICU).

In PICU, she required up to 2 inotropes for hypotension. In view of severe presentation with no significant history of ill contact, she was treated with meningitis. Intravenous cefotaxime and benzylpenicillin was given. However, her parents refused for lumbar puncture. Her septic parameters showed thrombocytosis with normal CRP. There was also no reported positive culture.Insulin infusion was continued with hourly dextrostix. She required addition of dextrose solution up to 12.5 % for hypoglycaemia. 6hrly arterial blood gas, blood urea and serum electrolyte, and urine ketone were monitored. She was out of DKA state with improving metabolic acidosis and followed by complete eradication of ketone bodies after 48 hours of admission.

Case report (cont)

The patient was extubated and also able to be wean off her inotrope support after 2 days. Her dextrostix ranged 6-8 mmol/l with insulin infusion of 0.01unit/kg/hr.The patient was transitioned with subcutaneous actrapid and the dose adjusted with the aim of dextrostix ranging 4-12 mmol/L. She was on breastfeeding on demand with topped up oral feeding 60mls/3hours. She was sent to endocrine team in Putrajaya with subcutaneous actrapid of 0.7/0.5/0.5 unit TDS for optimisation and further management. Her total admission to PICU was 16 days.

Her HbA1c, serum c-peptide, serum anti glutamic acid decarboxylase, anti-islet antibody and insulin autoantibodies were still pending. Her initial thyroid function test showed low T4 (6.89 pmol/L) and low TSH (0.188 mIU/L). Treatment was not started as there was a possibility of euthyroid sick syndrome. Her repeated TFT was normal with TSH 4.578mIU/L and FT4 21.52 pmol/L.

Discussion

The biochemical criteria for diagnosis of DKA are a venous pH <7.3 or serum bicarbonate concentration <15 mmol/L, serum glucose concentration more than 11 mmol/L, and ketonemia or moderate to large ketonuria. The severity of DKA is determined by the degree of acidosis. It is mild when venous pH 7.2– 7.3, moderate when pH 7.1–7.2 and severe when pH less than 7.1.[5,6,10]

DKA may presents with nausea and/ or vomiting, abdominal pain, blurry vision and confusion. On examination, patient may appear dehydrated, drowsy, deep sighing respiration and breath smells of acetone. Tachycardia, tachypnoea, drowsiness, progressive decrease in level of consciousness and, eventually loss of consciousness also among the signs of DKA.[5,6] All these presentations may be difficult to recognise in an infant if the possibility of DKA not even entertained, putting the patients at increased risk for delays in diagnosis. Which may lead to higher blood glucose levels and even severe DKA at presentation.[11,2] It is shown in a descriptive cohort study in Southern India from the year 1999 to 2012. Out of 67% of infant presented with DKA at onset, only 48% were initially diagnosed to have either diabetes or diabetic ketoacidosis.[1]

Our patient presented at the age of 54 days with hyperglycemia, severe acidosis and ketonemia consistent with the diagnosis of DKA.[5,6] Infection in the infant causes physiological stress and lead to counterregulatory hormone excess. These factors augment glucose production from glycogenolysis and gluconeogenesis. Hyperglycemia with limited glucose utilization (due to insulin deficiency) resulting in osmotic diuresis, electrolyte loss, dehydration, decreased glomerular filtration and hyperosmolarity .[3,5]

This patient was treated according to International Society for Paediatric and Adolescent Diabetes[6] and Malaysian CPG(Management of type 1 Diabetes Mellitus in children and adolescents). She was given fluid boluses, as she came in shock and 10 % correction given over 48 hrs along with fluid maintenance. Subsequently, she was started on insulin infusion 0.1 unit/kg with dextrostix monitoring. She was also treated for meningitis. A major difference between DKA in adult and paediatric is that children are particularly at risk for cerebral edema (CE).[9] Incidence of 11.9 per 1000 episodes was recorded in a study conducted through the British Paediatric Surveillance Unit between October 1995 and September 1998 with a significant mortality (24%) and morbidity (35% of surviviors).[12] For this reason, rapid fluid administration is discouraged. Total fluid deficits should be replaced over a 48-hour period.[9,16]

The exact cause of DKA-related CE is unknown, but it is likely that there are many factors involved. Children presenting with more severe DKA are at greatest risk. Among the risk factors are severe acidosis with higher blood urea nitrogen levels and hypocapnoea. Additional risk factors identified were early administration of insulin, high volumes of fluid and the rate of initial fluid administration. Hydrating at a rate greater than 50 mL/kg during the first 4 hours was associated with increased risk of brain herniation.[13-16]

Other complications in children with DKA include hyper- or hypokalemia with arrhythmias, sepsis, acute respiratory distress syndrome, pneumonia, pulmonary edema, rhabdomyolysis, and alveolar rupture.[9]

Conclusions

In conclusion, diabetic ketoacidosis is the most common presentation of infantile diabetes. Although infantile DKA is rare, it is life threatening and requires a high degree of suspicion especially in infant with coexisting infection. Any ill-looking, dehydrated infant should be screened for hyperglycemia since any physiological stress may lead to DKA in a child with previously undiagnosed diabetes mellitus.

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